

Physical treatment modalities

The use of cold and superficial heat in the treatment of soft tissue injuries

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Introduction

Over the years there has been much confusion about the use of ice in the treatment of acute and chronic injuries. Therapists claim to use ice to decrease circulation in the treatment of an acute injury yet claim the opposite in the treatment of chronic injury. The confusion stems from the report by Lewis in 1930 of the 'hunting response' of the circulation to tissue cooling¹. This, plus the easily observable reactive hyperaemia caused by cooling the skin, promoted the belief that the cooling of tissue caused the blood supply to decrease and then increase. Lewis concluded, from skin temperature measurements, that the blood flow continued to increase and decrease in a cyclical manner.

These opinions continue to be quoted in spite of a paper subsequently published by Abramson² and others which identified the cyclical response to cooling to occur at 60 per cent of the resting level of blood supply. Abramson concluded that the reduction in blood supply occurred in the underlying muscle. Cobbold and Lewis³ reported a similar reduction in blood flow following cooling of knee joints in dogs.

Therapeutic cold

In the immediate care of soft tissue injuries the reduction of tissue metabolism by cooling is probably more important than the reduction of blood supply. The oxygen consumption of the forearm immersed in water at 17°C significantly reduced oxygen consumption compared to resting values⁴. This effect of cooling was first used as an adjunct to surgery in the 1950s.

Jozsa⁵ described the time-dependent changes in muscle tissue following blunt trauma resulting from road traffic accidents. Myofilament damage reached its maximum two hours after the injury, with hypercontraction peripheral to the focus of injury possibly acting as a contributory factor. Cellular damage and cell death continued over the next 22 hours due to anoxia caused by oedema. Secondary cell death is self-perpetuating in that it is a source of more free protein leading to further oedema, more anoxia, and therefore more cell death. It would appear prudent to apply cold within the first two hours after injury to counter

hypercontraction, reduce tissue metabolism, and therefore minimize secondary cell death.

Further evidence to support the use of ice in acute injury was reported by Farry *et al.* in 1980⁶. Indirectly traumatized pig radiocarpal ligaments showed significantly less inflammatory response following cooling. However, subcutaneous swelling was increased. Dowart *et al.*⁷ reported similar findings for chemically-induced inflammation in knee joints, but xenon clearance rates from the joints were slowed. This supports the view that ice has no beneficial effect on joint swelling once the intra-articular effusion has formed.

McMaster *et al.*⁸ showed that ice packs reduce subcutaneous temperature more than chemical ice packs or gel packs. Crushed ice, or ice cubes with a small quantity of water, contained in a plastic bag is most convenient and does not cause frostbite in healthy adults⁹. An application of between 20 and 30 minutes duration will cause muscle temperatures to remain below normal for two hours at which time ice should be reapplied for a further 20 minutes. The importance of accompanying elevation of the limb cannot be underestimated¹¹.

The use of ice, exercise and gait re-education has been shown to be superior to exercise and gait re-education alone in the treatment of ankle sprains¹². In the sub-acute stage, the role of ice is to reduce pain to allow more vigorous pain-free exercise. Unlike pain killing injections, cold does not abolish sensitivity to pain which remains a protection against over vigorous exercise. Thus it appears that exercise rather than ice aids recovery¹³.

A comparative study of heat and cold treatments applied within the first 36 hours of ankle injuries showed that cold reduced time to recovery by eight days when compared to early heat treatment or the delayed application of cold (started 36 hours after injury)¹⁴. Similar claims have been made for the treatment of muscle injury.

Therapeutic heat

The benefits of heat are more obvious in chronic inflammation although care is needed because heating tissue causes a mild inflammatory reaction. Joint stiffness is decreased by surface heating techniques when

applied to superficial joints¹⁵. Temperatures of 6°C above normal levels have been recorded in foot joints following 20 minutes heating at 39°C in a water bath¹⁶. Heating connective tissues prior to application of longitudinal stretching aids permanent elongation of tissue without the evidence of tissue damage¹⁷. Heat must be applied for thirty minutes to raise the temperature of the tissues at a depth of one to two centimetres when using superficial heating agents like hot packs.

Hot packs are contained in an absorbent canvas bag which, like hot water bottles, avoids the skin maceration which results from repeated immersion in hot water. A greater increase in tissue temperature can be produced up to a depth of 1.2 centimetres by superficial heating modalities than by ultrasound or diathermy¹⁶. Superficial adipose tissue retards heat conduction.

Heat is important in the prevention of injury. The games player can reduce the risk of injury during cold wintry conditions by wearing thermal vests and leggings to conserve body heat. Cooling increases the muscle relaxation time¹⁸ and therefore increases the risks of antagonist muscles being disrupted by agonists during cyclical activity such as running. On the other hand, too great an increase in muscle temperature, such as that produced by immersion in a hot bath, decreases muscle force production¹⁹.

Although the effects of ice, exercise and conductive heating remain equivocal, they have important advantages. They are readily available to all and, after instruction, can be used independently of the therapist, although some supervision is advisable.

References

- 1 Lewis, T. Observations upon the reactions of the vessels of the human skin to cold *Heart* 1930, **15**, 177–208
- 2 Abramson, D.I., Chu, L.S.W., Tuck, S., Lee, S.W., Richardson, G. and Levin, M. Effect of tissue temperature and blood flow on motor nerve conduction velocity *JAMA* 1966, **198**, 1082–1088
- 3 Cobbald, A.F. and Lewis, O.J. Blood flow to the knee joint of a dog. Effect of heating, cooling, and adrenaline *J Physiol* 1956, **132**, 379–383
- 4 Abramson, D.I., Kahn, A., Tuck, S., Turman, G.A., Rejal, H. and Fleisher, C.J. Relationship between a range of tissue temperature and local oxygen consumption in the resting forearm *Lab Clin Med* 1957, **59**, 789–793
- 5 Jozsa, L. Fine structural study of human skeletal muscle injuries due to blunt trauma *J Legal Med* (Berlin) 1978, **82**(2), 145–152
- 6 Farry, P.J., Prentice, N.G., Hunter, A.C. and Wakelin, C.A. Ice treatment of injured ligaments: an experimental model *NZ Med J* 1980, **91**, 12–14
- 7 Dowart, B.D., Hansell, J.R. and Schumacher, H.R. Effect of cold and heat on urate crystal-induced synovitis in the dog *Arthritis and Rheumatism* 1974, **17**(5) 563–571
- 8 McMaster, W.C., Little, S. and Waugh, T.R. Laboratory evaluations of various cold therapy modalities *Am J Sports Med* 1978, **6**, 291–294
- 9 Knight, K.L. 'Cryotherapy theory, technique and physiology' 1st ed Chattanooga Corporation, Educational Division, Chattanooga, 1986
- 10 Hartviksen, K. Ice therapy in spasticity *Acta Neurol Scandinav* 1962, **38** (suppl 3) 140, 79–84
- 11 Sims, D. Effects of position on ankle oedema *J Orth and Sports Phys Ther* 1986, **8**, (1) 30–33
- 12 Knight, K.L. Ankle rehabilitation with cryotherapy *Phys Sportsmed* 1979, **7**(11) 133–137
- 13 Knight, K.L. and Londree, B.R. Comparison of blood flow in the ankle of uninjured subjects during therapeutic heat, cold and exercise *Med Sci Sport and Exercise* 1980, **12**, (1) 76–80
- 14 Hocutt, J.E. Cryotherapy in ankle sprains *Am J Sports Med* 1982, **10**, 316–319
- 15 Wright, V. and Johns, R.J. Physical factors concerned with the stiffness of normal and diseased joints *Bull Johns Hopkins Hosp* 1960, **106**, 215
- 16 Borrell, R.M. Comparison of in vivo temperatures produced by hydrotherapy, paraffin wax and fluidotherapy *Phys Ther* 1980, **60**, 1273–1276
- 17 Warren, G.C., Lehmann, J.F. and Koblanski, J.N. 1976. Heat and stretch procedures: an evaluation using rat tail tendon *Arch Phys Med Rehabil* 1976, **57**(3) 122–125
- 18 Edwards, R.H.T., Young, A., Hoskins, G.P. and Jones, D.A. Human skeletal muscle function: description of tests and normal values *Clin Sci and Mol Med* 1977, **52**, 283–290
- 19 Edwards, H.T. Effect of temperature on muscle energy metabolism and endurance during successive isometric contractions, sustained to fatigue, of quadriceps muscle in man *J Physiol* 1972, **220**, 335–341